Dated: May 12, 2016.

### Richard U. Rodriguez,

Associate Director, Technology Transfer Center, National Cancer Institute.

[FR Doc. 2016-11661 Filed 5-17-16; 8:45 am]

BILLING CODE 4140-01-P

# DEPARTMENT OF HEALTH AND HUMAN SERVICES

### **National Institutes of Health**

## National Institute of Dental & Craniofacial Research; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Dental and Craniofacial Research Special Emphasis Panel; NIDCR Data Analysis and Statistical Methodology PARs.

Date: June 10, 2016.

Time: 11:00 a.m. to 4:00 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, One Democracy Plaza, 6701 Democracy Boulevard, Bethesda, MD 20892.

Contact Person: Victor Henriquez, Ph.D., Scientific Review Officer DEA/SRB/NIDCR, 6701 Democracy Blvd., Room 668, Bethesda, MD 20892–4878, 301–451–2405, henriquv@nidcr.nih.gov.

 $\it Name\ of\ Committee:\ NIDCR\ Special\ Grants$  Review Committee.

Date: June 16-17, 2016.

Health, HHS)

Time: 8:00 a.m. to 12:00 p.m.

Agenda: To review and evaluate grant applications.

Place: Wyndham San Antonio Riverwalk 111 East Pecan Street, San Antonio, TX 78205

Contact Person: Marilyn Moore-Hoon, Ph.D., Scientific Review Officer, Scientific Review Branch, National Institute of Dental and Craniofacial Research, 6701 Democracy Blvd., Rm. 676, Bethesda, MD 20892–4878, 301–594–4861, mooremar@nidcr.nih.gov. (Catalogue of Federal Domestic Assistance Program Nos. 93.121, Oral Diseases and Disorders Research, National Institutes of

Dated: May 12, 2016.

## Natasha M. Copeland,

Program Analyst, Office of Federal Advisory Committee Policy.

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# DEPARTMENT OF HEALTH AND HUMAN SERVICES

### **National Institutes of Health**

Prospective Grant of an Exclusive License: The Development of an Anti-GPC3 Chimeric Antigen Receptor (CAR) Based on YP7 for the Treatment of Human Cancers

**AGENCY:** Public Health Service, National Institutes of Health, HHS.

**ACTION:** Notice.

**SUMMARY:** This notice, in accordance with 35 U.S.C. 209(c)(1) and 37 CFR Part 404.7(a)(1)(i), that the National Institutes of Health, Department of Health and Human Services, is contemplating the grant of an exclusive license to practice the inventions embodied in:

## **Intellectual Property**

U.S. Provisional Patent Application 61/654,232 entitled "High-affinity Monoclonal Antibodies To Glypican-3 And Use Thereof" [HHS Ref. E-136-2012/0–US–01]; PCT Patent Application PCT/US2013/043633 entitled "Highaffinity Monoclonal Antibodies To Glypican-3 And Use Thereof" [HHS Ref. E-136-2012/0-PCT-02]; Chinese Patent Application 201380039993.7 entitled "High-affinity Monoclonal Antibodies To Glypican-3 And Use Thereof' [HHS Ref. E-136-2012/0-CN-03]; Japanese Patent Application 2015–515243 entitled "High-affinity Monoclonal Antibodies To Glypican-3 And Use Thereof" [HHS Ref. E-136-2012/0-JP-04]; South Korea Patent Application 10-2014-7037046 entitled "High-affinity Monoclonal Antibodies To Glypican-3 And Use Thereof" [HHS Ref. Ĕ-136-2012/0-KR-05]; Singapore Patent Application 11201407972R entitled "High-affinity Monoclonal Antibodies To Glypican-3 And Use Thereof" [HHS Ref. E-136-2012/0-SG-06]; United States Patent Application 14/403,896 entitled "High-affinity Monoclonal Antibodies To Glypican-3 And Use Thereof" [HHS Ref. E-136-2012/0-US-

and all continuing U.S. and foreign patents/patent applications for the technology family, to Lentigen Technology, Inc.

The patent rights to these inventions have been assigned to and/or

exclusively licensed to the Government of the United States of America.

The prospective exclusive licensed territory may be the United States, Australia, Canada, the European Union, Russia, China, Hong Kong, Japan, Taiwan, South Korea and Singapore, and the field of use may be limited to: "The development of a glypican-3 (GPC3) chimeric antigen receptor (CAR)based immunotherapy using autologous (meaning one individual is both the donor and the recipient) primary human lymphocytes (T cells or NK cells) transfected with a lentiviral or retroviral vector, wherein the vector expresses a CAR having (1) a single antigen specificity and (2) comprising at least: (a) The complementary determining region (CDR) sequences of the anti-GPC3 antibody known as YP7; and (b) a T cell signaling domain; for the prophylaxis and treatment of GPC3-expressing cancers."

**DATES:** Only written comments and/or applications for a license which are received by the NCI Technology Transfer Center on or before June 2, 2016 will be considered.

ADDRESSEES: Requests for copies of the patent application, inquiries, comments, and other materials relating to the contemplated exclusive license should be directed to: David A. Lambertson, Ph.D., Senior Licensing and Patenting Manager, National Cancer Institute, 9609 Medical Center Drive, Rm 1–E530 MSC9702, Rockville, MD 20850–9702, Email: david.lambertson@nih.gov.

**SUPPLEMENTARY INFORMATION:** This invention concerns an anti-GPC3 (Glypican-3) chimeric antigen receptor (CAR) and methods of using the CAR for the treatment of GPC3-expressing cancers. GPC3 is a cell surface antigen that is preferentially expressed on certain types of cancer cells, particularly liver cancers such as hepatocellular carcinoma (HCC). The anti-GPC3 CARs of this technology contain (1) antigen recognition sequences that bind specifically to GPC3 and (2) signaling domains that can activate the cytotoxic functions of a T cell. The anti-GPC3 CAR can be transduced into T cells that are harvested from a donor, followed by (a) selection and expansion of the T cells expressing the anti-GPC3 CAR, and (b) reintroduction of the T cells into the patient. Once the anti-GPC3 CARexpressing T cells are reintroduced into the patient, the T cells can selectively bind to GPC3-expressing cancer cells through its antigen recognition sequences, thereby activating the T cell through its signaling domains to selectively kill the cancer cells. Through this mechanism of action, the selectivity